Changes in sheep oesophageal diameter and function during Geigeria ornativa (vermeerbos) poisoning and subsequent recovery

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ABSTRACT

Changes in the oesophageal diameter and function together with changes in body weight, feed intake and the cardiac pulmonary flow index were investigated during experimentally induced poisoning with Geigeria ornativa and subsequent recovery. This was performed under varying conditions for individual sheep. Results showed an increase in the oesophageal diameter index (ODI) during vermeersiekte, accompanied with a decrease in oesophageal function (OF). Cessation of G. ornativa intake resulted in a considerable although incomplete recovery of the ODI. Recovery of the OF for the different sheep, however, varied between 0 and 100 %. Detrimental changes in the oesophageal diameter and function were also measured in sheep receiving only subclinical doses of *G. ornativa*. Decreases in body weight and feed intake commenced 1 to 3 weeks before the onset of vermeersiekte, while indications of a decline in these 2 parameters were also noticed with ingestion of subclinical amounts of G. ornativa. An increase in the cardiac pulmonary flow index (CPFI) to a value indicating the onset of heart failure was found in 1 of the sheep showing clinical signs of vermeersiekte. The CPFI returned to normal after termination of *G. ornativa* intake.

Key words: body weight, cardiac pulmonary flow index, feed intake, Geigeria ornativa poisoning, oesophageal diameter, oesophageal function, vermeersiekte.

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INTRODUCTION

Geigeria ornativa poisoning induces vermeersiekte, one of the most important plant poisonings of sheep on the subcontinent². It is characterised by regurgitation of ruminal contents through the mouth and nose, as well as stiffness, paresis and paralysis³. At necropsy dilatation of the oesophagus is sometimes evident3. The prognosis of vermeersiekte is usually good if stock is promptly removed from the toxic grazing^{1,3}. However, Van Heerden, Van der Lugt & Durante (1993)⁷ reported that mild dilatation of the oesophagus was still radiographically evident 55 days after intake of *G. ornativa*. This may have an impairing effect on the oesophageal function, resulting in decreased feed intake and production. Changes in the oesophageal diameter and function during and following clinical and subclinical intoxication with *G. ornativa* were investigated in case studies with sheep.

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METHODS AND RESULTS

The effect of different doses of G. ornativa on the oesophageal diameter and function, body weight (BW), feed intake and the cardiopulmonary flow index (CPFI) for measuring cardiac function⁶ was studied in 7 adult sheep. Sheep were presented with 20-100 g milled G. ornativa mixed

with 100 g maize meal and 200 g milled lucerne each morning. They additionally received milled Eragrostis curvula hay ad lib. daily after the G. ornativa mixture had been ingested, which took between 1 and 2 hours to consume. Remains of the previous day's Eragrostis curvula hay were weighed before feeding.

Body weight was determined weekly in the morning after sheep had been fasted overnight.

The oesophageal diameter, expressed in terms of the oesophageal diameter index (ODI) (oesophageal diameter as a ratio of the length of the 7th thoracic vertebral body, illustrated in Fig. 1)⁴, was measured by survey radiography at maximal width. This was measured prior to any positive contrast medium administration and could only be done when there was luminal oesophageal gas present, which was the case in most of the affected animals. Oesophageal function (OF) was measured with fluoroscopy by following a swallowed bolus of milled feed mixed with barium contrast medium4. If a feed bolus passed directly through the cardiac sphincter into the reticulorumen, the OF was regarded as normal and quantified as 100 % functional. If the bolus did not pass directly into the reticulorumen, but lingered for a short time until a secondary or tertiary peristaltic wave allowed it to

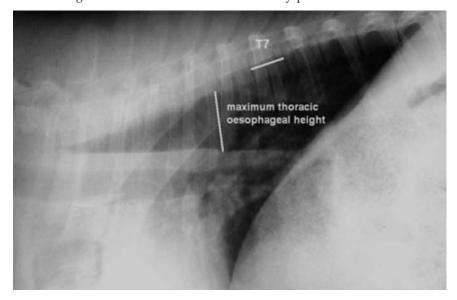


Fig. 1: Survey radiograph: maximum oesophageal height; length of the 7th thoracic vertebral body.

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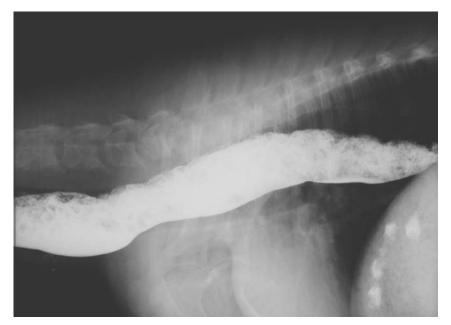


Fig. 2: Post-barium-meal radiograph: accumulation of food material in the thoracic oesophagus of a sheep extremely affected by vermeersiekte.

move through, the OF was considered as moderately affected and arbitrarily quantified as 75 % functional. If feed was partially retained in the thoracic oesophagus, the OF was described as severely affected and arbitrarily quantified as 50 % functional. If the swallowed feed accumulated in the thoracic oesophagus, as illustrated in Fig. 2, and did not move aborally at all during the examination time of a few minutes, the OF was described as extremely affected and arbitrarily quantified as 25 % functional (the OF was not quantified as 0 % since these sheep still

managed to eat the forage presented to them).

The CPFI was obtained by measuring the number of heart beats necessary to pump radioactive technetium (^{99m}Tc pertechnetate) in the blood from the heart to the lungs and back⁶.

Values for the different parameters, expressed as percentage of the maximum value, were graphically presented for each sheep.

Sheep 1, a Dorper wether, was fed *G. ornativa* collected in the Griekwastad (28°51′10″S, 23°14′54″E) district, Northern

Cape. The dried plant material, mainly leafy, was fed for 42 days at a dose of 3.2 g/kg BW/day. The effect of G. ornativa during feeding and following its withdrawal on the different parameters until Day 202 is shown in Fig. 3. Recovery of the oesophageal diameter and function after withdrawal from G. ornativa until Day 776 is shown in Figs 8a and 8b respectively. The sheep revealed clinical signs of stiffness and paresis from Day 27 to Day 49. The oesophageal diameter index increased exceptionally during the occurrence of vermeersiekte, accompanied by c. 50 % decrease in oesophageal function. Body weight and feed intake were also adversely affected. Except for the oesophageal function, recovery of these parameters commenced soon after cessation of G. ornativa intake. Incomplete recovery of the oesophageal diameter index, with no recovery of the oesophageal function, was measured over a period of 776 days following cessation of vermeerbos intake.

Sheep 2, a Merino wether, was fed with the same *G. ornativa* as Sheep 1, but at a dose of 2 g/kg BW/day for 25 days. Values for the different parameters measured until Day 79 are given in Fig. 4a. Signs of pronounced stiffness, paresis, and a green staining around the nose (indicating some regurgitation of rumen contents) became evident from Day 21 to 32. In addition to detrimental changes of the oesophageal diameter index and function, hay intake and body weight, the cardiac pulmonary flow index increased to a

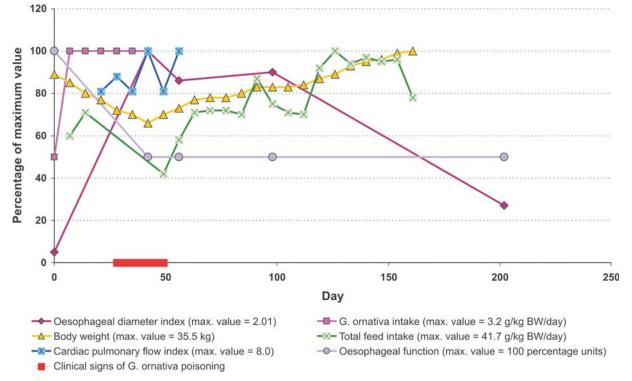
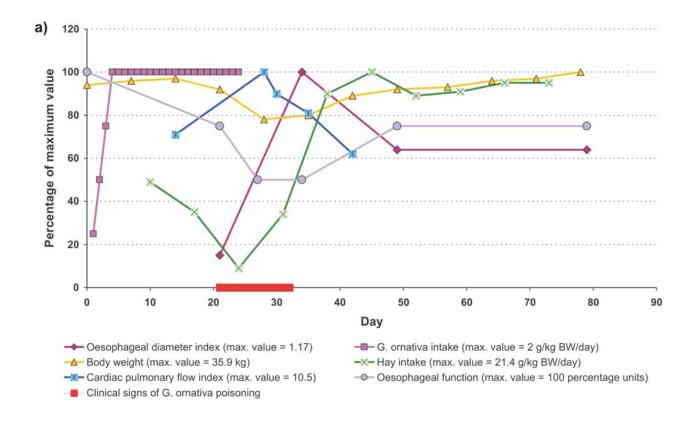


Fig. 3: Changes in some clinical and physiological parameters of Sheep 1 during poisoning with *Geigeria ornativa* and subsequent recovery.



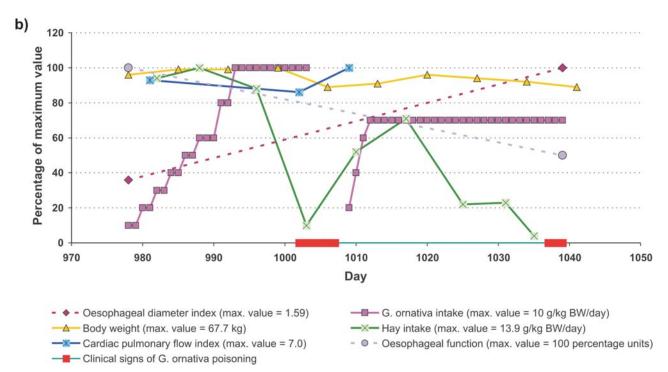


Fig. 4: a: Changes in some clinical and physiological parameters of Sheep 2 during poisoning with *Geigeria ornativa* and subsequent recovery. b: Changes in some clinical and physiological parameters of Sheep 2 during a 2nd and 3rd intake of *G. ornativa*.

value suggesting poor heart function. All these parameters showed some recovery after ingestion of *G. ornativa* was stopped. The oesophageal diameter index and function after withdrawal from *G. ornativa* until Day 978 are shown in Figs 8a and 8b, respectively. The data show full recovery of the oesophageal function but incomplete recovery of the oesophageal diameter index.

On Day 978 and Day 1009 the sheep was exposed again during 2 successive periods (Days 978–1003 and Days 1009–1039) with apparently low-toxicity *G. ornativa* collected on another farm in the Griekwastad district. Results are shown in Fig. 4b. The sheep developed clinical signs of vermeersiekte, showing paresis, regurgitation of rumen contents (staining around the nose), and some stiffness, from Day

1002 to Day 1008 and again from Day 1037 to 1039. Body weight and hay intake tended to decrease during ingestion of *G. ornativa*, resulting in vermeersiekte, followed by recovery after withdrawal from *G. ornativa*. The oesophageal diameter index increased and the oesophageal function decreased over the total period.

Sheep 3, a Van Rooyen–Persian cross breed wether, was also fed with the

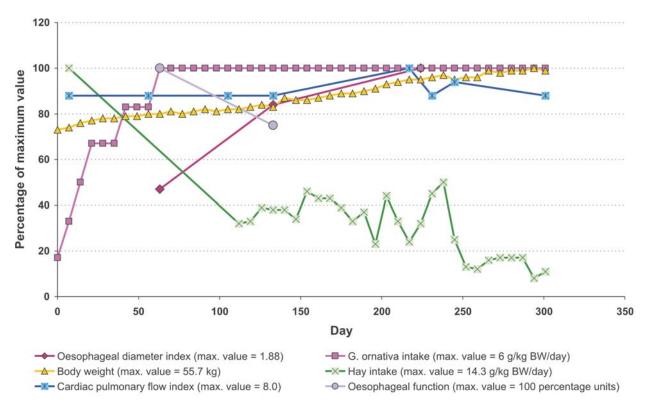


Fig. 5: Changes in some clinical and physiological parameters of Sheep 3 during poisoning with Geigeria ornativa.

apparently low-toxicity *G. ornativa*. Intake of the milled plant material during the first 63 days was gradually increased from 1 g/kg BW/day to 6 g/kg BW/day and kept at that level until Day 301. Results are shown in Fig. 5. The sheep never showed clinical signs of vermeersiekte but had some difficulty in swallowing the *G. ornativa*-containing mixture from Day 161. A slight green staining in the nose was evident on Day 224 and during the

end of the feeding period it was noticed that the sheep intermittently drank water during ingestion of the *G. ornativa* mixture. He held his head upwards when swallowing the mixture. The oesophageal diameter index increased during the trial, while hay intake diminished. A decrease in the oesophageal function was also measured during the trial.

Sheep 4, a Dorper wether, was fed with the same *G. ornativa* given to Sheep 1 at a

dose of 1.2 g/kg BW/day for 91 days. Results are shown in Fig. 6. The sheep did not develop clinical signs of vermeersiekte. Total feed intake increased some time after cessation of vermeerbos intake, accompanied by a small increase in body weight.

Sheep 5, a Van Rooyen-Persian cross breed wether, received increasing doses of the apparently low-toxicity *G. ornativa*, namely: 1.5 g/kg BW/day (Days 0–7),

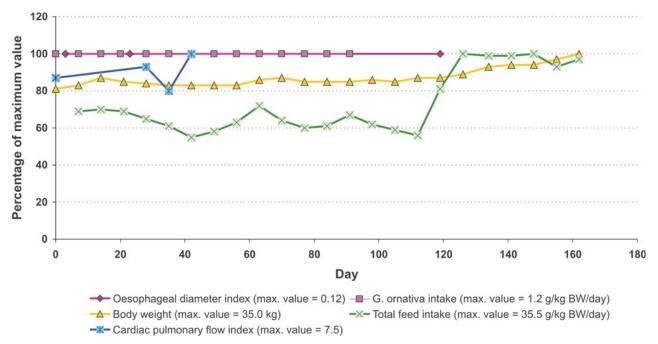


Fig. 6: Changes in some clinical and physiological parameters of Sheep 4 during poisoning with *Geigeria ornativa* and subsequent recovery.

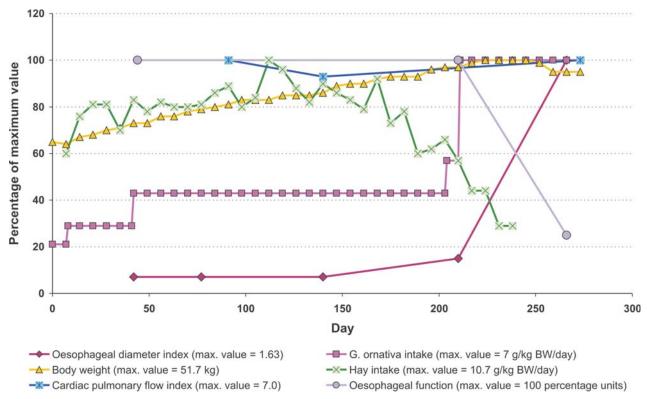


Fig. 7: Changes in some clinical and physiological parameters of Sheep 5 during poisoning with Geigeria ornativa.

2.0 g/kg BW/day (Days 8–41), 3.0 g/kg BW/day (Days 42–203), 4.0 g/kg BW/day (Days 204–210) and 7 g/kg BW/day (Days 211–273). Results are shown in Fig. 7. No clinical signs of vermeersiekte were observed. The oesophageal diameter index and function remained almost unchanged until the last increase in *G. ornativa* intake, when both of the parameters changed drastically. The increase in body weight that took place up to this stage levelled out, while hay intake continued the decrease that started from an earlier stage of the trial.

Sheep 6 and 7 (Dorper ewes) were obtained at the end of February 2000 from a farmer in the Griekwastad district. According to the farmer, the sheep had been removed from vermeerbos-infested veld more than once after developing clinical signs reminiscent of vermeersiekte (regurgitation of rumen contents). After withdrawal from the veld (Day 0) they were kept in a pen at the Onderstepoort Veterinary Institute and the oesophageal diameter and function monitored over time. The sheep were fed milled *E. curvula* hay ad lib., supplemented with approximately 200 g pelleted concentrate per day. Changes in the oesophageal diameter and function of Sheep 6 are presented in Figs 8a and 8b, respectively. Incomplete recovery of the oesophageal diameter and function took place during this period of more than 3 years.

Sheep 7 stopped ingesting hay 30 days after cessation of *G. ornativa* intake. Clinical examination of the sheep revealed that

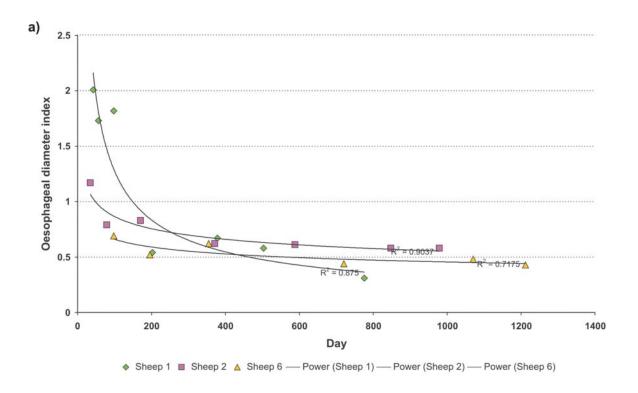
the oesophagus was packed with ingesta, preventing ingestion of hay. Mechanical removal of the oesophageal contents allowed the sheep to eat and drink again. Measurement of the oesophageal parameters on Day 34 showed an ODI of 2.04 and an extremely weakened oesophageal function.

OBSERVATIONS AND CONCLUSIONS

The following observations were made from the results of this investigation:

- Clinical signs of vermeersiekte developed after 21 to 28 days of G. ornativa intake at doses of 2 and 3.2 g/kg BW/day (Sheep 1 and 2, Figs 3 and 4a). A 5-day cessation of G. ornativa intake resulted in clinical recovery from vermeersiekte (Sheep 1 and 2; Figs 3, 4a and 4b). G. ornativa then had to be ingested for another 27 days (a lower dose) before clinical signs of vermeersiekte could be re-induced (Sheep 2, Fig. 4b). Vermeersiekte, however, could not be induced when G. ornativa was fed at levels varying between 30 % and 70 % of the corresponding toxic doses (Sheep 3, 4 and 5, Figs 5, 6 and 7), indicating the possibility of a threshold dose that has to be over-
- A dilated oesophagus with concomitant weakened oesophageal function developed in all cases of vermeersiekte (Sheep 1, 2, 6 and 7; Figs 3, 4a, 4b, 8a and 8b). Extreme dilatation of the oesophagus after repeated occurrence of vermeersiekte on *G. ornativa* infested pasture resulted in complete oesophageal dys-

- function (Sheep 7). Deteriorating oesophageal changes did not only occur with clinical vermeersiekte, as pronounced dilatation of the oesophagus with a decrease in oesophageal function was also noticed in a sheep receiving only a sub-clinical dose of *G. ornativa* (Sheep 3 and 5; Figs 5 and 7).
- Reduced feed intake and a loss in body weight preceded clinical signs of vermeersiekte by approximately 7 to 21 days (Sheep 1 and 2; Figs 3, 4a and 4b). Indications of reduced growth rate and feed intake, however, were also noticed for sheep receiving sub-clinical doses of *G. ornativa* (Sheep 3, 4 and 5; Figs 5, 6 and 7).
- Decreased cardiac function in sheep suffering from vermeersiekte was demonstrated for the first time by measuring the CPFI (Sheep 2, Fig. 4a) (A CPFI value of c. 7 reflects normal heart function while a value of c. 10 indicates the onset of heart failure⁶). This is consistent with histological damage demonstrated in the heart muscle of sheep with vermeersiekte⁵ and with an earlier report¹ listing cardiac failure as one of the causes of death in affected sheep. A decrease in cardiac function, however, could not be demonstrated in a sheep that received an even higher dose of the same batch of G. ornativa (Sheep 1, Fig. 3).
- Recovery from vermeersiekte occurred fairly rapidly, as clinical signs disappeared within 5–7 days after termination of *G. ornativa* intake (Sheep 1 and 2, Figs 3, 4a and 4b). Cessation of *G. orna-*



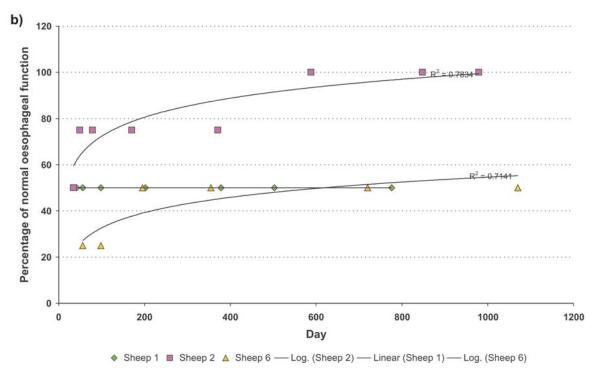


Fig. 8: a: Changes in oesophageal diameter index values of sheep during long-term recovery from vermeersiekte. b: Changes in oesophageal function of sheep during long-term recovery from vermeersiekte.

tiva intake was followed shortly by an increase in body weight and feed intake (Figs 3, 4a and 4b). A considerable, although incomplete, recovery of the oesophageal diameter occurred (normal ODI values c. 0.1) after cessation of G. ornativa intake (Fig. 8a). Recovery was characterised by an initial rapid recovery that slowed down with time. Recovery of the oesophageal function

varied from full to no recovery at all (Fig. 8b). The increased CPFI during vermeersiekte returned to normal within 17 days after withdrawal from *G. ornativa,* indicating that the decrease in cardiac function was a reversible process that normalised quite rapidly.

• These observations might be of great significance for optimal flock management on *G. ornativa* infested veld and

therefore need to be confirmed in a well designed study.

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